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=> d his

     (FILE 'HOME' ENTERED AT 13:05:26 ON 13 MAR 2008)

     FILE 'CAPLUS' ENTERED AT 13:05:39 ON 13 MAR 2008
L1       197631 S ESR OR EPR OR (ELECTRON (5A) SPECTROSCOPY)
L2      1204560 S RADIAT? OR IRRADIAT?
L3        36856 S ASCORBATE
L4        58454 S UVA OR (UV (2A) A)

=> s l1 and l2 and l3 and l4
L5           12 L1 AND L2 AND L3 AND L4

=> d ti 1-12

L5   ANSWER 1 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Protein, lipid, and DNA radicals to measure skin UVA damage and
     modulation by melanin

L5   ANSWER 2 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Relevance of sunscreen application method, visible light, and sunlight
     intensity to free-radical protection: a study of ex vivo human skin

L5   ANSWER 3 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Thioridazine induces immediate and delayed erythema in photopatch test

L5   ANSWER 4 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   A comparison of UV-B induced stress responses in three
     barley cultivars

L5   ANSWER 5 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Reduction of UVB/A-generated free radicals by sodium L-ascorbyl-2-
     phosphate in cultured mouse skin

L5   ANSWER 6 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   The inhibitory effects of UV-B radiation (280-315 nm) on Gunnera
     magellanica growth correlate with increased DNA damage but not with
     oxidative damage to lipids

L5   ANSWER 7 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Inorganic ionic molecular crystal

L5   ANSWER 8 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Method and apparatus for determining effectiveness of sunscreens and other
     skin preparations in shielding human skin from UVA
     radiation

L5   ANSWER 9 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   EPR detection of free radicals in UV-irradiated skin:


L5   ANSWER 10 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Inhibitory effect of sodium 5,6-benzylidene ascorbate (SBA) on
     the elevation of melanin biosynthesis induced by ultraviolet-A (
     UV-A) light in cultured B-16 melanoma cells

L5   ANSWER 11 OF 12  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Ultraviolet light-induced free radical formation in skin: an electron
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paramagnetic resonance study

L5 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI Spectral and paramagnetic properties of oxyhemoglobin solutions exposed to UV-radiation in the presence of ascorbic acid

=> s skin
275445 SKIN
10870 SKINS
L6 281554 SKIN
(SKIN OR SKINS)

=> s l5 and l6
L7 6 L5 AND L6

=> d ti 1-6

L7 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
TI Protein, lipid, and DNA radicals to measure skin UVA damage and modulation by melanin

L7 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
TI Relevance of sunscreen application method, visible light, and sunlight intensity to free-radical protection: a study of ex vivo human skin

L7 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
TI Reduction of UVB/A-generated free radicals by sodium L-ascorbyl-2-phosphate in cultured mouse skin

L7 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method and apparatus for determining effectiveness of sunscreens and other skin preparations in shielding human skin from UVA radiation

L7 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
TI EPR detection of free radicals in UV-irradiated skin: mouse versus human

L7 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
TI Ultraviolet light-induced free radical formation in skin: an electron paramagnetic resonance study

=> d ibib abs hit 1-6

L7 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:304151 CAPLUS <<LOGINID::20080313>>
TITLE: Protein, lipid, and DNA radicals to measure skin UVA damage and modulation by melanin
AUTHOR(S): Haywood, Rachel; Rogge, Fabrice; Lee, Martin
CORPORATE SOURCE: RAFT Institute of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex, HA6 2RN
SOURCE: Free Radical Biology & Medicine (2008), 44(6), 990-1000
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Afro-Caribbeans have a lower incidence of skin cancer than Caucasians, but the effectiveness of melanin as a photoprotective pigment is debated. We investigated the UVA and solar irradiation of ex vivo human skin and DMPO using ESR spectroscopy, to determine whether pigmented skin is protected by melanin against free radical damage. Initial ascorbate radicals in Caucasian skin were superseded by lipid and/or protein radical adducts with isotropic (a(H) = 1.8 mT) and anisotropic spectra comparable to spectra in irradiated pig fat (a(H) = 1.9 mT) and BSA. DNA carbon-centered radical adducts (a(H) = 2.3 mT) and a broad singlet were detected in genomic DNA/melanin but were not distinguishable in irradiated Caucasian skin. Protein and lipid radicals (n = 6 in Caucasian skin) were minimal in Afro-Caribbean skin (n = 4) and intermediate skin pigmentations were variable (n = 3). In irradiated Afro-Caribbean skin a shoulder to the melanin radical (also in UVA-irradiated pigmented melanoma cells and genomic DNA/melanin and intrinsic to pheomelanin) was detected. In this sample group, protein (but not lipid) radical adducts decreased directly with pigmentation. ESR/spin trapping methodol. has potential for screening skin susceptibility to aging and cancer-related radical damage and for measuring protection afforded by melanin, sunscreens, and antiaging creams.
TI Protein, lipid, and DNA radicals to measure skin UVA damage and modulation by melanin
AB Afro-Caribbeans have a lower incidence of skin cancer than Caucasians, but the effectiveness of melanin as a photoprotective pigment is debated. We investigated the UVA and solar irradiation of ex vivo human skin and DMPO using ESR spectroscopy, to determine whether pigmented skin is protected by melanin against free radical damage. Initial ascorbate radicals in Caucasian skin were superseded by lipid and/or protein radical adducts with isotropic (a(H) = 1.8 mT) and anisotropic spectra comparable to spectra in irradiated pig fat (a(H) = 1.9 mT) and BSA. DNA carbon-centered radical adducts (a(H) = 2.3 mT) and a broad singlet were detected in genomic DNA/melanin but were not distinguishable in irradiated Caucasian skin. Protein and lipid radicals (n = 6 in Caucasian skin) were minimal in Afro-Caribbean skin (n = 4) and intermediate skin pigmentations were variable (n = 3). In irradiated Afro-Caribbean skin a shoulder to the melanin radical (also in UVA-irradiated pigmented melanoma cells and genomic DNA/melanin and intrinsic to pheomelanin) was detected. In this sample group, protein (but not lipid) radical adducts decreased directly with pigmentation. ESR/spin trapping methodol. has potential for screening skin susceptibility to aging and cancer-related radical damage and for measuring protection afforded by melanin, sunscreens, and antiaging creams.

L7 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:953398 CAPLUS <<LOGINID::20080313>>
DOCUMENT NUMBER: 145:403428
TITLE: Relevance of sunscreen application method, visible light, and sunlight intensity to free-radical protection: a study of ex vivo human skin
AUTHOR(S): Haywood, Rachel
CORPORATE SOURCE: Mount Vernon Hospital, RAFT Institute of Plastic Surgery, Northwood, Middlesex, UK
SOURCE: Photochemistry and Photobiology (2006), 82(4),

1123-1131
CODEN: PHCBAP; ISSN: 0031-8655
PUBLISHER: American Society for Photobiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB With the continued rise in skin cancers worldwide there is a need for effective skin protection against sunlight damage. It was shown previously that sunscreens, which claimed UVA protection (SPF 20+), provided limited protection against UV-induced ascorbate radicals in human skin. Here the results of an ESR investigation to irradiate ex vivo human skin with solar-simulated light are reported. The ascorbate radical signal in the majority of skin samples was directly proportional to the irradiance over relevant sunlight intensities (0.9-2.9 mW cm-2). Radical production (substratum-corneum) by UV (wavelengths <400 nm) and visible components (>400 nm) was .apprx.67 and 33% resp. Ascorbate radicals were in steady state concentration at low irradiance (.apprx.1 mW cm-2 equivalent to UK sunlight), but at higher irradiance (.apprx.3 mW cm-2) decreased with time, suggesting ascorbate depletion. Radical protection by a 4 star-rated sunscreen (with UVA protection) was optimal when applied as a thin film (40-60% at 2 mg cm-2) but less so when rubbed into the skin (37% at 4 mg cm-2 and no significant protection at 2 mg cm-2), possibly due to cream filling crevices, which reduced film thickness. This study validates ESR detns. of the ascorbate radical for quant. protection measurements. Visible light contribution to radical production, and loss of protection when sunscreen is rubbed into skin, has implications for sunscreen design and use for the prevention of free-radical damage.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Relevance of sunscreen application method, visible light, and sunlight intensity to free-radical protection: a study of ex vivo human skin

AB With the continued rise in skin cancers worldwide there is a need for effective skin protection against sunlight damage. It was shown previously that sunscreens, which claimed UVA protection (SPF 20+), provided limited protection against UV-induced ascorbate radicals in human skin. Here the results of an ESR investigation to irradiate ex vivo human skin with solar-simulated light are reported. The ascorbate radical signal in the majority of skin samples was directly proportional to the irradiance over relevant sunlight intensities (0.9-2.9 mW cm-2). Radical production (substratum-corneum) by UV (wavelengths <400 nm) and visible components (>400 nm) was .apprx.67 and 33% resp. Ascorbate radicals were in steady state concentration at low irradiance (.apprx.1 mW cm-2 equivalent to UK sunlight), but at higher irradiance (.apprx.3 mW cm-2) decreased with time, suggesting ascorbate depletion. Radical protection by a 4 star-rated sunscreen (with UVA protection) was optimal when applied as a thin film (40-60% at 2 mg cm-2) but less so when rubbed into the skin (37% at 4 mg cm-2 and no significant protection at 2 mg cm-2), possibly due to cream filling crevices, which reduced film thickness. This study validates ESR detns. of the ascorbate radical for quant. protection measurements. Visible light contribution to radical production, and loss of protection when sunscreen is rubbed into skin, has implications for sunscreen design and use for the prevention of free-radical damage.

ST sunscreen solar irradn light radical protection skin

IT Solar radiation
(IR; sunscreen application method, visible light, and sunlight intensity to free-radical protection)
IT IR radiation
(solar; sunscreen application method, visible light, and sunlight intensity to free-radical protection)
IT Human
Light
Skin
Solar radiation
Sunscreens
(sunscreen application method, visible light, and sunlight intensity to free-radical protection)

L7 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:417280 CAPLUS <<LOGINID::20080313>>
DOCUMENT NUMBER: 144:66007
TITLE: Reduction of UVB/A-generated free radicals by sodium L-ascorbyl-2-phosphate in cultured mouse skin
AUTHOR(S): Masatsuji-Kato, Eiko; Tsuzuki, Toshi; Kobayashi, Shizuko
CORPORATE SOURCE: Corp. R & D Cent., Showa Denko K. K., Chiba, 267-0056, Japan
SOURCE: Journal of Health Science (2005), 51(2), 122-129
CODEN: JHSCFD; ISSN: 1344-9702
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The quenching abilities of sodium L-ascorbyl-2-phosphate (APS) and ascorbic acid 2-glucose (AG) against UVB/A-generated free radicals in cultured mouse skin were investigated using ESR. The relation between their quenching ability and protective effects against photodamage were also compared to those of ascorbic acid (AsA) pretreatment. Both APS and AG were able to scavenge UVB/A-generated hydroxyl radicals under aqueous conditions (pH 7.2) in a manner similar to that seen with AsA; however, APS was a more effective scavenger than AG. Similar results were obtained ex vivo. Both derivs. could protect skin from UVB/A-induced photodamage, as determined by a reduction in the presence of sunburn cells and DNA fragmentation. However, AsA pretreatment had the weakest protective effect, even though cutaneous, its level was the highest among the three agents tested before irradn . These results indicated that the superior protective effect of APS is related to its direct free radical scavenging ability, rather than to its conversion to AsA.
REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Reduction of UVB/A-generated free radicals by sodium L-ascorbyl-2-phosphate in cultured mouse skin
AB The quenching abilities of sodium L-ascorbyl-2-phosphate (APS) and ascorbic acid 2-glucose (AG) against UVB/A-generated free radicals in cultured mouse skin were investigated using ESR. The relation between their quenching ability and protective effects against photodamage were also compared to those of ascorbic acid (AsA) pretreatment. Both APS and AG were able to scavenge UVB/A-generated hydroxyl radicals under aqueous conditions (pH 7.2) in a manner similar to that seen with AsA; however, APS was a more effective scavenger than AG. Similar results were obtained ex vivo. Both derivs. could protect skin from UVB/A-induced photodamage, as determined by a reduction in the presence of sunburn cells and DNA fragmentation. However, AsA

pretreatment had the weakest protective effect, even though cutaneous, its level was the highest among the three agents tested before irradn . These results indicated that the superior protective effect of APS is related to its direct free radical scavenging ability, rather than to its conversion to AsA.

ST sodium ascorbyl phosphate UV radical scavenger; ascorbate deriv UV radiation skin radical scavenger

IT Injury
(cutaneous; reduction of UVB/A-generated free radicals by ascorbic acid derivs. including sodium L-ascorbyl-2-phosphate in cultured mouse skin)

IT Skin, disease
(injury; reduction of UVB/A-generated free radicals by ascorbic acid derivs. including sodium L-ascorbyl-2-phosphate in cultured mouse skin)

IT Photoprotectants
Radical scavengers
UV A radiation
UV B radiation
(reduction of UVB/A-generated free radicals by ascorbic acid derivs. including sodium L-ascorbyl-2-phosphate in cultured mouse skin)

IT Reactive oxygen species
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reduction of UVB/A-generated free radicals by ascorbic acid derivs. including sodium L-ascorbyl-2-phosphate in cultured mouse skin)

IT 3352-57-6, Hydroxy radical, biological studies 7782-44-7D, Oxygen, reactive species 109620-90-8 562043-82-7
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reduction of UVB/A-generated free radicals by ascorbic acid derivs. including sodium L-ascorbyl-2-phosphate in cultured mouse skin)

L7 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:387291 CAPLUS <<LOGINID::20080313>>
DOCUMENT NUMBER: 140:380275
TITLE: Method and apparatus for determining effectiveness of sunscreens and other skin preparations in shielding human skin from UVA radiation
INVENTOR(S): Haywood, Rachel Mary; Wardman, Peter; Sanders, Roy; Linge, Claire
PATENT ASSIGNEE(S): Raft Trustees Ltd., UK
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004039414	A1	20040513	WO 2003-GB4637	20031028

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM,

TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2504346	A1	20040513	CA 2003-2504346	20031028
AU 2003278353	A1	20040525	AU 2003-278353	20031028
EP 1590003	A1	20051102	EP 2003-769664	20031028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 2006133996	A1	20060622	US 2005-533617	20051104
PRIORITY APPLN. INFO.:			GB 2002-25408 A	20021031
			WO 2003-GB4637 W	20031028

AB The present invention provides a method and apparatus for measuring the effectiveness of a sunscreen composition or other skin preparation in reducing the exposure of human skin to UVA radiation using differential ESR spectroscopy to quantify the extent of UVA-induced ascorbate or other measurable radical production in the shielded skin, in comparison with reference, preferably unshielded, skin.

TI Method and apparatus for determining effectiveness of sunscreens and other skin preparations in shielding human skin from UVA radiation

AB The present invention provides a method and apparatus for measuring the effectiveness of a sunscreen composition or other skin preparation in reducing the exposure of human skin to UVA radiation using differential ESR spectroscopy to quantify the extent of UVA-induced ascorbate or other measurable radical production in the shielded skin, in comparison with reference, preferably unshielded, skin.

ST radical ESR spectroscopy sunscreen skin UVA radiation; ascorbate ESR spectroscopy sunscreen skin UVA radiation

IT ESR spectroscopy
Human
Skin
Sunscreens
UV A radiation
(ESR spectroscopy for measurement of radical production in determining effectiveness of sunscreens and other skin prepns. in shielding human skin from UVA radiation)

IT Radicals, analysis
RL: ANT (Analyte); FMU (Formation, unclassified); ANST (Analytical study); FORM (Formation, nonpreparative)
(ESR spectroscopy for measurement of radical production in determining effectiveness of sunscreens and other skin prepns. in shielding human skin from UVA radiation)

IT 299-36-5, Ascorbate, analysis
RL: ANT (Analyte); FMU (Formation, unclassified); ANST (Analytical study); FORM (Formation, nonpreparative)
(ESR spectroscopy for measurement of radical production in determining effectiveness of sunscreens and other skin prepns. in shielding human skin from UVA radiation)

L7 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:942 CAPLUS <<LOGINID::20080313>>
DOCUMENT NUMBER: 126:115134
TITLE: EPR detection of free radicals in UV-irradiated skin: mouse versus human

AUTHOR(S): Jurkiewicz, Beth Anne; Buettner, Garry R.
CORPORATE SOURCE: Free Radical Res. Inst., Univ. Iowa, Iowa City, IA, USA
SOURCE: Photochemistry and Photobiology (1996), 64(6), 918-922
CODEN: PHCBAP; ISSN: 0031-8655
PUBLISHER: American Society for Photobiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB UV radiation produces free radicals in Skh-1 mouse skin , contributing to photoaging and carcinogenesis. If a mouse model is a general indicator of free radical processes in human skin photobiol., then radical production observed in mouse and human skin should be directly comparative. In this work we show that UV radiation (λ > 300 nm, 14 μW/cm2 UVB; 3.5 mW/cm2 UVA) increases the ascorbate free radical (Asc.-) ESR (EPR) signal in both Skh-1 mouse skin (45%) and human facial skin biopsies (340%). Visible light (λ > 400 nm; 0.23 mW/cm2 UVA) also increased the Asc.- signal in human skin samples (45%) but did not increase baseline mouse Asc.-, indicating that human skin is more susceptible to free radical formation and that a chromophore for visible light may be present. Using EPR spin-trapping techniques, UV radiation produced spin adducts consistent with trapping lipid alkyl radicals in mouse skin (α-[4-pyridyl 1-oxide]-N-tert-Bu nitrone/alkyl radical adduct; aN = 15.56 G and aH = 2.70 G) and lipid alkoxyl radicals in human skin (5,5-dimethylpyrroline-1-oxide/alkoxyl radical adduct; aN = 14.54 G and aH = 16.0 G). Topical application of the iron chelator Desferal to human skin significantly decreases these radicals (≈50%), indicating a role for iron in lipid peroxidn.; Desferal has previously been shown to decrease radical production in mouse skin. This work supports the use of the Skh-1 mouse as a predictive tool for free radical formation in human skin. These results provide the first direct evidence for UV radiation-induced free radical formation at near physiol. temps. in human skin and suggest that iron chelators may be useful as photoprotective agents.
TI EPR detection of free radicals in UV-irradiated skin: mouse versus human
AB UV radiation produces free radicals in Skh-1 mouse skin , contributing to photoaging and carcinogenesis. If a mouse model is a general indicator of free radical processes in human skin photobiol., then radical production observed in mouse and human skin should be directly comparative. In this work we show that UV radiation (λ > 300 nm, 14 μW/cm2 UVB; 3.5 mW/cm2 UVA) increases the ascorbate free radical (Asc.-) ESR (EPR) signal in both Skh-1 mouse skin (45%) and human facial skin biopsies (340%). Visible light (λ > 400 nm; 0.23 mW/cm2 UVA) also increased the Asc.- signal in human skin samples (45%) but did not increase baseline mouse Asc.-, indicating that human skin is more susceptible to free radical formation and that a chromophore for visible light may be present. Using EPR spin-trapping techniques, UV radiation produced spin adducts consistent with trapping lipid alkyl radicals in mouse skin (α-[4-pyridyl 1-oxide]-N-tert-Bu nitrone/alkyl radical adduct; aN = 15.56 G and aH = 2.70 G) and lipid alkoxyl radicals in human skin (5,5-dimethylpyrroline-1-oxide/alkoxyl radical adduct; aN = 14.54 G and aH = 16.0 G). Topical application of the iron chelator Desferal to human skin significantly decreases these radicals (≈50%),

indicating a role for iron in lipid peroxidn.; Desferal has previously been shown to decrease radical production in mouse skin. This work supports the use of the Skh-1 mouse as a predictive tool for free radical formation in human skin. These results provide the first direct evidence for UV radiation-induced free radical formation at near physiol. temps. in human skin and suggest that iron chelators may be useful as photoprotective agents.

ST UV radiation radical mouse skin model; iron chelator desferal photoprotectant UV radiation

IT Light
Photoprotectants
Skin
UV radiation
(UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT Radicals, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT Alcohols, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(aliphatic, radicals; UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT Chelating agents
(iron; UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT Peroxidation
(lipid; UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT Lipids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(peroxidn.; UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT Mouse
(skin, model; UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT 138-14-7, Desferal
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

IT 6730-29-6, Ascorbate radical, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(UV radiation-induced free radical formation in mouse vs.

human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)
IT 7439-89-6, Iron, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study) (UV radiation-induced free radical formation in mouse vs. human skin in relation to mouse model use, iron role in lipid peroxidn., and iron chelators use as photoprotectants)

L7 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1994:157602 CAPLUS <<LOGINID::20080313>>
DOCUMENT NUMBER: 120:157602
TITLE: Ultraviolet light-induced free radical formation in skin: an electron paramagnetic resonance study
AUTHOR(S): Jurkiewicz, Beth Anne; Buettner, Garry R.
CORPORATE SOURCE: Coll. Med., Univ. Iowa, Iowa City, IA, 52242-1101, USA
SOURCE: Photochemistry and Photobiology (1994), 59(1), 1-4
CODEN: PHCBAP; ISSN: 0031-8655
DOCUMENT TYPE: Journal
LANGUAGE: English
AB It has been suggested that UV light induces free radical formation in skin, leading to photoaging and cancer. The authors have demonstrated by ESR that the ascorbate free radical is naturally present in unexposed skin at a very low steady state level. When a section of SKH-1 hairless mouse skin in an EPR cavity is exposed to UV light (4500 J/m2/s, Xe lamp, 305 nm cutoff and IR filters), the ascorbate free radical signal intensity increases. These results indicate that UV light increases free radical oxidative stress, consistent with ascorbate's role as the terminal, small-mol. antioxidant. The initial radicals produced by UV light would have very short lifetimes at room temperature; thus, the authors have applied EPR spin trapping techniques to detect these radicals. Using α-[4-pyridyl 1-oxide]-N-tert-Bu nitrone (POBN), the authors have for the first time spin trapped a UV light-produced carbon-centered free radical from intact skin. The EPR spectra exhibited hyperfine splittings that are characteristic of POBN/alkyl radicals, aN = 15.56 G and aH = 2.70 G, possibly generated from membrane lipids as a result of β-scission of lipid alkoxyl radicals. Iron can act as a catalyst for free radical oxidative reactions.; chronic exposure of skin to UV radiation causes increased iron deposition. Using the authors' spin trapping system, the authors have shown that topical application of the iron-chelator, Desferal, to a section of skin reduces the UV light-induced POBN adduct radical signal. These results provide direct evidence for free radical generation and a role for iron in UV light-induced dermatopathol. The authors suggest that iron chelators can serve as photoprotective agents by preventing these oxidns.
TI Ultraviolet light-induced free radical formation in skin: an electron paramagnetic resonance study
AB It has been suggested that UV light induces free radical formation in skin, leading to photoaging and cancer. The authors have demonstrated by ESR that the ascorbate free radical is naturally present in unexposed skin at a very low steady state level. When a section of SKH-1 hairless mouse skin in an EPR cavity is exposed to UV light (4500 J/m2/s, Xe lamp, 305 nm cutoff and IR filters), the ascorbate free radical signal intensity increases. These results indicate that UV light increases free radical oxidative stress, consistent with ascorbate's role as the terminal, small-mol. antioxidant. The initial radicals produced by UV light would have very short lifetimes at room temperature; thus, the authors

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ST UV skin radical formation

IT Skin, metabolism
(UV radiation-induced radical formation in)

IT Radicals, biological studies
RL: FORM (Formation, nonpreparative)
(formation of, in skin, UV radiation induction of)

IT Ultraviolet radiation
(radical formation in skin induced by)

IT 6730-29-6, Ascorbate radical, biological studies
RL: FORM (Formation, nonpreparative)
(formation of, in skin, UV radiation induction of)